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Provocative Testing for Embolization of Spinal Cord AVMs

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Summary

The purpose of this study is to evaluate efficacy and reliability of chemical provocative testing using neurophysiological monitoring prior to embolization of spinal cord AVMs (SCAVMs). We performed retrospective analysis of provocative testing using sodium amytal and lidocaine injected superselectively in 41 angiography and / or embolization procedures in 26 patients with a SCAVM, including 23 amytal and 26 lidocaine injections. All procedures were performed under general anesthesia using neuroleptic drugs, and with monitoring of cortical somatosensory evoked potentials (SEPs) and trans-cranial motor evoked potentials (MEPs). After recording baseline SEPs and MEPs, 50mg of sodium amytal was injected through the microcatheter at the position of the intended embolization, followed by assessment of SEPs and MEPs. If no changes occurred, 40mg of lidocaine was then injected followed by recording of SEPs and MEPs. If again no changes were noted, embolization was performed using NBCA. If there was any change in either SEPs or MEPs, NBCA embolization was not performed from that catheter position. No false negative results of the provocative testing were experienced. One amytal test from the posterior spinal artery (PSA) was positive, causing loss of MEPs. Lidocaine testing was positive in 10 cases including 4 injections in the PSA (with loss of MEPs in two and SEPs in two), 5 injections in the anterior spinal artery (with loss of MEPs in four and SEPs in one), and 1 case involving the posterior inferior cerebellar artery (with loss of MEPs). Neither amytal nor lidocaine injection caused loss of both SEPs and MEPs. In conclusion, sodium amytal and lidocaine are complimentary as pharmacological agents for provocative testing, and SEPs and MEPs are complimentary to each other as physiologic monitoring methods. Provocative testing should be performed using both amytal and lidocaine with monitoring of both SEPs and MEPs.

Introduction

When embolizing spinal cord AVMs, it is imperative to preserve the blood supply to the normal spinal cord. Angiographic identification of spinal cord arteries may be difficult, despite magnification or additional lateral and oblique views, due to their small size and overlapping normal and pathological vasculature. In addition, due to the hemodynamic changes produced by the spinal cord AVM, the normal spinal cord supply may not be predicted based solely on angiographic findings.

In addition to the careful angiographical analysis, clinical provocative testing is a method used to identify the functional eloquence of the territory of a catheterized vessel. It is performed by clinically assessing neurological

changes after the injection of a short acting anesthetic from a microcatheter which has been placed in an feeding artery for the AVM [1]. However, for spinal cord AVM embolization procedures, we prefer general anesthesia to control patient breathing in order to obtain high-resolution images to identify small spinal cord vessels. For the cases performed under general anesthesia, electrophysiological monitoring rather than clinical assessment is used for provocative testing.

Initially only cortical somatosensory evoked potentials (SEPs) were monitored [2], but its reliability in assessing the corticospinal tract was not ideal [3]. Therefore, we started using motor evoked potentials (MEPs) in addition to SEPs. One of major problems of previously reported technique in monitoring MEPs was its invasiveness such as making a burr hole or inserting an epidural electrode [4]. One of us (VD) has set up a protocol for a non-invasive technique of transcranial cortical stimulation along with recording from peripheral muscles [5,6,7]. We are presenting our experience with SCAVM embolization using this monitoring technique.

Material and Methods

The patient is maintained under general anesthesia using propofol (100-150μ / Kg / min) and fentanyl (1μg / Kg / hr) drip. No gaseous anesthetics or muscle relaxants are used except for during anesthesia induction.

SEP-monitoring was performed in a conventional method [8]. Briefly, SEPs were elicited by stimulating the posterior tibial and median nerves on each side by using electric current (40 mA, 0.2 ms, 4.3 Hz). SEPs were recorded via corkscrew-like electrodes placed on the scalp over the sensory cortex. MEPs were elicited with transcranial electrical stimulation of the motor cortex using corkscrew type electrodes. Short trains of 5-7 square-wave stimuli of 500 μs duration and 4 ms inter-stimulus interval were applied at 1 Hz frequency through electrodes placed at C1 and C2 scalp sites according to the International 10 / 20 Electroencephalogram System. The intensity of stimulation did not exceed 160 mA. Muscle responses were recorded from needle electrodes inserted into both anterior tibialis and thenar muscles [6] (figure 1). Following recording of the baseline SEPs and MEPs and prior to embolization, provocative testing for neuronal function was performed by injection of 50 mg of sodium amytal from the microcatheter placed in a feeder of the AVM at the location intended for embolization. If there were no changes in SEPs or MEPs, this was followed by injection of 20-40 mg of lidocaine depending on the feeder size and degree of shunting. If there were still no changes in SEPs or MEPs, embolization was performed using N-butyl cyanoacrylate (NBCA) from that catheter position. If any change in the SEPs or MEPs after injection of sodium amytal or lidocaine occurred, the provocative test was considered positive and NBCA embolization from that catheter position was not performed.

The results of SEPs and MEPs monitoring and provocative tests in 41 angiography / embolization procedures for SCAVMs performed since 1996 were retrospectively analyzed.

Results

SEPs and MEPs monitoring was attempted in all 41 procedures. It was possible to obtain monitorable SEPs in 85% and MEPs in 97% of cases. Twenty-three amytal and 26 lidocaine tests were performed with one (5%) positive amytal test and 10 (37%) positive lidocaine tests. The summary of the positive tests is shown in table 1. The positive sodium amytal test occurred during an injection of a posterior spinal artery (PSA) feeder with loss of MEPs. The positive lidocaine tests included 4 injections in the PSA (loss of MEP in 2 and SEPs in 2); 5 injections in the anterior spinal artery (ASA) (loss of MEPs in 4 and SEPs in one) and one injection of the posterior inferior cerebellar artery with loss of MEPs. There was no catheterized vessel in which both sodium amytal and lidocaine tests were positive. In addition, a positive injection resulted in changes in either SEPs or MEPs but not both. No patient showed worsening of the symptoms after embolization using this provocative test method (i.e. there was no false negative results).

Discussion

In the early 1980s, we began solely monitoring SEPs during spinal cord angiography and embolization procedures with relatively good reliability [1,2]. Although there were several re-

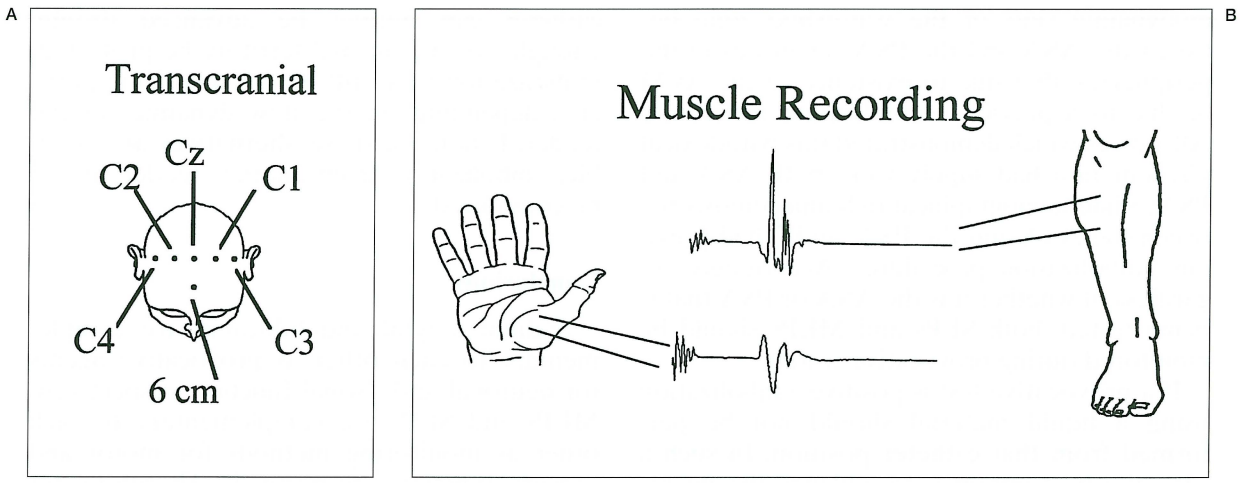

Figure 1

ports that SEPs monitoring failed to predict postoperative motor deficits [3,9], MEPs monitoring during embolization was underutilized due to the necessity of invasive burr holes and insertion of epidural electrodes [4]. Due to these concerns, a protocol for reliable non-invasive monitoring of MEPs [7] was established in the mid-1990s by one of the senior authors (V.D.).

In our series of treated patients, we used pharmacological provocative testing prior to planned embolization in order to minimize the risk of neurological complications. Sodium amytal is a short-acting barbiturate that predominantly suppresses neuronal activity as opposed to lidocaine which predominantly suppresses axonal conduction in the central nervous system [10]. Due to their complementary modes of neural inhibition, we have favored the use of both agents during provocative testing to best identify vascular territories at risk for dysfunction after embolization. This theory of selective inhibition is supported by our observation that only one agent (and not the other) resulted in suppression of evoked potentials when injected into a vessel of interest, but suppression never occurred with both agents in a particular vessel. Similarly, regardless of the pharmaceutical agent used, there was no tested vessel that demonstrated suppression on both SEPs and MEPs monitoring.

It is interesting to note that one injection of sodium amytal in the PSA and in 2 injections of lidocaine in the PSA resulted in loss of MEPs without changes in SEPs and one injection of lidocaine in the ASA resulted in loss of SEPs without changes in MEPs. These observations are contradictory to the general understanding that the ASA supplies the anteriorly located motor pathway and the PSA supplies the posteriorly located sensory pathway of the proprioception, which is most important for elicitation of SEPs. These phenomena suggest that the territory of the ASA or the PSA can be infused by superselective injection of anesthetics through either the PSA or the ASA, respectively. This may be due to the rich anastomosis between the ASA and the PSA territories either through the nidus of the AVM itself or through peri-nidal anastomoses. There may also be he-

Table 1 **Summary of positive results of provocative testing**

Agent	Vessel studied	Change in:	No. of cases
Amytal	PSA	MEP	1
Lidocaine	PSA	MEP	2
		SEP	2
	ASA	MEP	4
		SEP	1
	PICA	MEP	1
Total			11

PSA = Posterior spinal artery; ASA = Anterior Spinal Artery; PICA = Posterior Inferior Cerebellar Artery; SEP = Cortical Somatosensory Evoked Potential; MEP = Motor Evoked Potential

modynamic shift of the watershed zone between the ASA and the PSA territories in the periphery, either due to existence of the AVM or due to a previous embolization procedure. All 4 cases which demonstrated this paradoxical phenomenon had supply from both ASA and PSA; 3 had angiographical rich anastomosis between the ASA and the PSA, and 2 had previous embolization procedures. Accordingly, regardless of whether it is the ASA or PSA that is being tested, both SEPs and MEPs should be monitored during provocative testing.

If a provocative test is positive, embolization using a liquid material should not be performed from that catheter position. In such a case, the best option is to advance the microcatheter closer to the nidus. Another option is to protect the normal territory using a fiber or liquid coil. If, after blocking this territory, the repeated provocative test is negative, liquid embolization can then be performed. If the catheter can neither be advanced distally enough, nor the normal territory be protected, embolization may still be possible using particles, depending on the flow dynamics of the feeder. If none of these alternatives are possible, embolization from another feeder should be considered.

Conclusions

Sodium amytal and lidocaine are complementary to each other as provocative agents for neuronal and axonal function, respectively. MEPs and SEPs are complementary to each other as monitoring methods for motor and sensory functions, respectively. Therefore, for the safer embolization of spinal cord AVMs under general anesthesia, provocative tests should be performed using both sodium amytal and lidocaine and both MEPs and CSEPs should be monitored.

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